TREATMENT BY THE TOURNIQUET TO COUNTERACT THE VASOMOTOR SPASM OF RAYNAUD'S DISEASE.

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I have been privileged by Dr. Osler to report from his clinic the following case. The object of doing so is to call attention to a form of treatment that apparently proved beneficial in relieving a cycle of vasomotor overaction which in this particular instance had provoked a painful and protracted condition of "local asphyxia" of the four extremities.

Since Raynaud's final contribution in 1874[1] to the subject of the disease bearing his name there has been no particular advance in our knowledge of the physiological or pathological states underlying it. Its physiological basis seems to rest on the overaction in constriction of the arterioles of certain vascular territories. This overaction may be brought about by slight peripheral stimuli, which only under the abnormal conditions of the malady could produce reflexly such an exaggeration of the customary vaso-constrictor response. Thus a minimal lowering of the surface temperature for example may suffice in some cases to inaugurate an attack characterized by one type or another of the symptoms assumed by the disease. In order to account for the characteristically symmetrical nature of the vascular disturbance so often observed, Raynaud was led to believe in the existence of a lesion or state of overexcitability of the vasomotor centers themselves.[2]

Presumably the same fundamental condition, whatever it may be, is responsible—through variations in the intensity of its action—for the three well known gradations in severity of symptoms: the "local syncope" (Raynaud) with exsanguinated tissues: the "local asphyxia" from venous stasis, and finally the thrombosis and gangrene, representing the terminal feature of the vascular spasm in case the latter is sufficiently severe or prolonged in its action.

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[1] Archives générales de médecine T. I., Jan., 1874. Trans. by Barlow, New Sydenham Soc., 1888.

[2] "New Researches on the Nature and Treatment of Local Aspyhxia of the Extremities," *loc. cit.* p. 182. "To sum up in a more definite form, I would say that in the present state of our knowledge local asphyxia of the extremities ought to be considered as a neurosis characterized by enormous exaggeration of the excito-motor energy of the gray parts of the spinal cord which control the vasomotor innervation."

The opportunities offered for a post-mortem study of the disease have been rare and the information gained has been scant and unreliable. No change in the nervous system or in the blood vessels has been demonstrated in typical and uncomplicated cases. There is no endarteritis such as occasions the somewhat similar gangrenous processes in the senile; no change demonstrable in the nerves as in cases of symmetrical gangrene associated with peripheral neuritis. Cases of chilblains, frostbite and instances of ergot poisoning with prolonged constriction of arterioles leading to anemia, asphyxia and death of the extremities possibly present points of nearest resemblance to the conditions presented in Raynaud's disease.

Forty years have passed since the appearance of Raynaud's original thesis, and yet with the exception of one or two new points in symptomatology, our understanding of the malady remains where he left it. Barlow, in his recent chapter in Allbutt's System, says: "The earliest explanation of this pathological habit appears to be Raynaud's hypothesis; namely, the vasomotor center or centers are unduly irritable, that the commonest irritant is from the periphery, for example, cold; and that the different impulses from the center lead to the paroxysmal contraction of arterioles."

The suggestion that the application of a tourniquet to an extremity, early in the course of a period of arterial spasm, might temporarily throw out of action the nerves controlling the peripheral vasomotor mechanism and thus interrupt the syncopal attack, was based purely on this theory of physiological overaction of the neuro-vascular apparatus.

The removal of a tourniquet or Esmarch bandage, which has been applied to an extremity for operative purposes, is invariably followed by a flushing of the member, peripheral to the site of constriction, with bright arterial blood. From the embarrassment often occasioned thereby, this is a circumstance with which an operator is but too well acquainted.

This active hyperemia with increased surface temperature presumably is due to the temporary paralysis of vasomotor control to the part below the encircling bandage. Whether the pressure of the tourniquet produces a local compression paralysis or whether the resultant anemia throws the terminals of the vasomotor apparatus out of gear, it is impossible to say, but it suffices that the non-medullated fibers are "blocked" in consequence of the constriction by a more moderate and shorter application than is necessary to throw out of function those fibers conveying sensation and motion. Familiarity with the vasodilatation occurring under these circumstances naturally suggested the employment of a similar procedure as a therapeutic measure to counteract the local conditions of spasm characterizing the disease in question.

In the particular patient in whom this treatment was first instituted the symptoms were severe, the arterial spasm was pronounced and was associated with great pain. For several months a condition of local asphyxia of all the digits had been present with exacerbations of almost daily occurrence. On several occasions these attacks were so severe that slight superficial patches of terminal gangrene had affected the pads on one or more of the toes or fingers. The healing after this loss of substance had left most of the finger-tips tapering and firm. With the idea of interrupting the vaso-constrictor control over the individual digits as they became most actively involved, it was thought possible that cocainization of the lateral nerves might block the transmission of vasomotor as well as other nervous impulses and thus check the process.* This, however, was not tried as the tourniquet apparently accomplished the same purpose and controlled a wider vascular field. As is rather unusual the arterial spasm in this case extended far enough up the extremity to render the radial artery at times unpalpable at the wrist.

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*It is not impossible that a subarachnoid cocainization via the lumbar meninges might serve the purpose of abruptly throwing out of action the spinal vasomotor centers in these cases.

The history of the case is as follows:

The patient, Mrs. ———, 35 years of age, entered Dr. Osler's service October 29, 1900, complaining of "blueness and pains in the extremities." Her history, taken by Dr. Cole, shows no hereditary predisposition nor associated or previous illness, malaria, lues, diabetes, etc., which might have borne some relation to her present trouble.

She was never a strong child. She had scarlet fever, two attacks of diphtheria and other infantile infections. She suffered frequently during her early years from pains in her joints, usually worse during cold or damp weather. These pains have persisted more or less throughout her life. They have never been associated with evident articular changes nor have they been severe enough to incapacitate her for her usual duties. She has suffered also since childhood from attacks of "chilblains" which never required severe cold for their incitement. From this she has suffered every winter, though with advancing years there has been no especial increase in the severity of the attacks. She says that during the attacks the toes or fingers would become "red and swollen and would itch and burn."

She was married at 24 and became the mother of two children. Following the birth of one of them she was invalided for several months as the result of a severe infection. Her catamenial history is irregular. She suffers from frequent attacks of indigestion and vomiting which seem to bear no relation to indiscretions of diet. She has been more or less of an invalid for the past six years.

Although presumably akin to her former mild winter attacks, she dates the onset of her present serious trouble from March, 1901, several months before her admission to the hospital. Shortly after an attack of la grippe she began to be troubled with burning and itching of the legs and feet. One leg she says became red and swollen though the symptoms were most pronounced in the feet and toes. An upright position aggravated the condition and recumbancy became necessary. After a week or ten days the symptoms became associated with the extreme burning pain characteristic of cases of "local asphyxia."

In a more or less aggravated form, the arms and hands also having become affected, these symptoms had persisted uninfluenced by treatment during the ensuing months. There has been recourse to morphia almost daily to obtain some degree of relief from the pain and to induce sleep. On several occasions after a particularly severe exacerbation of the condition, a small, dry, brown eschar had formed on the pads of one or more fingers or toes.

She has had no symptoms referable to the viscera, to the central nervous system nor to vision.

Beyond the condition of the extremities, her physical examination was practically negative. She was a small, well nourished woman of neurotic temperament.

Dr. Osler's note on the extremities was as follows: "The hands are slightly congested, cold and moist. There is a slow return of vascularity after compression. The terminal phalanges are blue; the finger tips especially so. The fingers are tapering and the pads show dessicating epithelium and scars of old sloughs. The nails are rigid. On the outer side of the arms are scars where the skin has been involved in the last attack.

The arteries, neither radial nor brachial are palpable. Pulse is very small and feeble.

There is a general lividity of the toes and feet. The toes show a condition similar to the fingers though more advanced. There is considerable dessication of the skin, marked blueness of the tips and scars of old healed gangrenous sloughs. They are very painful and tender to manipulation. The tip of the nose is dark colored, deeply congested, but not swollen."

Blood Examination.—R.B.C., 4,800,000; W.B.C., 10,600; hemoglobin, 65 per cent.; coagulation time, one minute, twenty seconds.

Urine Examination showed nothing noteworthy. Almost daily observations over a period of two months showed no trace of hemoglobin.

With no evident amelioration, the patient's symptoms continued practically unaltered for the following two weeks. There was more or less constant discomfort with attacks of severe burning pain once or twice daily associated with blueness of the extremities. These exacerbations were only influenced by morphia. Although galvanism was not employed, the usual forms of treatment seemed to do little more than temporarily palliate the severity of the symptoms.

On November 10, during an exacerbation of pain and asphyxia chiefly affecting the left upper extremity, the flat rubber bandage was first employed as a tourniquet about the upper arm. Its application occasioned considerable local discomfort and pain referred to the side of the thorax (intercosto-humeral?). On its removal after one or two minutes' application a bright flush of the extremity followed with increase of surface temperature and a much more readily palpable radial artery. The vasomotor relaxation lasted only a short time, but the patient expressed a sensation of considerable relief. From this time seems to date the beginning of her improvement. The tourniquet was applied daily to one or another member according as the symptoms indicated. The local discomfort which it occasioned at first was subsequently less severe or was better endured and the application finally was extended to periods of five minutes or longer. The relief to the burning pain in the fingers was so pronounced that after the first few trials the patient was not only very willing to submit to the temporary discomfort of constriction, but would call for it.

An attempt was made to apply the tourniquet as early as possible in the period of exacerbation of pain and lividity. These periods occurred, however, with such irregularity that it was difficult or impossible to anticipate and ward off the vascular spasm by applying the tourniquet as a prophylactic measure, although an endeavor was made to do so. Improvement in the circulatory condition, however, was noticed almost from the first. The patient soon ceased to require morphia to induce sleep and the attacks became less and less frequent and of decreasing severity. After two or three weeks she was able to get up and move about her room without discomfort. Exposure from cold as from driving in the open air brought on no further trouble. She left the hospital apparently quite well on December 28, having gained ten pounds in weight, and having had for the preceding two weeks no recurrence of her distressing symptoms. The extremities at this time had resumed their original physiological tint.

During the patient's residence in the hospital while the recurring spasms with pain and lividity of the extremities were still severe, careful observations on the blood pressure were made by Drs. Cole and Erlanger. They failed, however, to demonstrate any characteristic rise in the general arterial tension other than that which could be accounted for by pain. It is probable that the vascular territories subjected to the vasoconstrictor action at any one time were too circumscribed to occasion a rise in the general blood pressure of any significance. A corresponding constriction of the splanchnic field would have produced a tremendous rise in arterial tension. A "local asphyxia" of the splanchnic venous system on the other hand, corresponding to the degree of stasis in the extremities, would have brought about a great fall in blood pressure. Apparently in this case, as heretofore stated, there were no circulatory disturbances referable to the viscera or central nervous system.

For the remainder of the winter months, following her discharge from the hospital, the patient remained free from symptoms and was able to resume her household duties with enjoyment. On one occasion in the spring, after fatigue and exposure to cold, she was threatened with a recurrence of the trouble. Once more her hands became livid in color and so painful that her attending physician administered morphia, but without giving her any relief. Recourse was made once more to the tourniquet with immediate improvement in the condition, and after a few applications the dreaded symptoms entirely disappeared. Since then up to the present writing she has had no return of trouble.

Galvanism in one form or another has proved of most benefit in the treatment of these conditions. This form of therapy is strongly advocated by Raynaud in his second essay, in which striking examples of improvement under its employment are cited. The rationale of the treatment depends upon the supposed relaxation of the overacting vasomotor centers in the cord consequent upon the passage of the electrical current.

The treatment by the tourniquet is most simple of application and from its apparent success in this instance may be possibly deserving of further trial. It depends seemingly in principle upon the physiological "blocking" effect of the elastic constriction on the peripheral vasomotor nerves. This temporary interruption of function allows the terminal arterioles to completely relax and a state of arterial hyperemia to take the place of the local asphyxia which has resulted from the pre-existing spasm. If employed early enough or repeated with sufficient frequency, as this case suggests, the hyper-excitability of the center, if that is the underlying cause of the spasm, either subsides or else, and what is more probable, the local vascular conditions in the extremities become so altered by the periods of active circulation that slight peripheral stimuli no longer provoke the intense reflex constrictor responses.

The treatment, of course, has its limitations, and is not applicable to a case where vascular spasm affects territories other than those confined to the limbs.